and California longshoremen (*3*) also provide extensive additional information about coronary heart disease in male cigarette smokers as compared to nonsmokers, supporting the above statements as they pertain to men.

The study of British physicians (*8*, *9*, *10*) suggests that male cigarette smokers have the largest increase in risk for death certified to coronary thrombosis—a subcategory of coronary heart disease describing acute coronary events, frequently occlusive, causing myocardial infarction. For that subcategory, the mortality ratio is also largest for the younger age groups 35–54.

Prospective morbidity studies confirm the relationships between cigarette smoking and coronary heart disease. These studies also provide the opportunity to evaluate the effect of smoking independently and in combination with other known "risk factors," such as high blood pressure and high serum cholesterol that are also important in the pathogenesis of coronary heart disease. It has been demonstrated that cigarette smoking not only operates as an independent "risk factor" but that it may combine with other "risk factors" to produce even greater effects on cardiovascular health.

Other types of evidence have also been presented to confirm the epidemiologic evidence. Autopsy studies show that cigarette smokers have a much greater frequency of advanced coronary arteriosclerosis than do nonsmokers. Clinical and experimental studies demonstrate that smoking produces abnormalities of cardiovascular physiology that may help to explain the mechanisms of how smoking may produce earlier death from coronary heart disease.

Human and experimental studies indicate that the nicotine absorbed from smoking may cause an increase in the myocardial tissue demand for oxygen yet at the same time the carbon monoxide absorbed from smoking may cause a decrease in the supply of available oxygen from the blood necessary to meet the increased myocardial tissue demand. Studies indicate that some persons who already have preexisting coronary heart disease, not necessarily clinically obvious, may be especially susceptible to the adverse physiological effects of smoking. Evidence also indicates that important differences may exist between normal individuals and those with coronary heart disease in their ability to increase coronary blood flow to compensate for increased myocardial tissue oxygen demand. Smoking apparently can accelerate thrombus formation of human blood, suggesting another possible mechanism whereby smoking might increase the mortality from coronary heart disease, especially those acute coronary events certified as "coronary thrombosis."

The convergence of many types of evidence—epidemiological, experimental, pathological, and clinical—strongly suggests that cigarette smoking can cause death from coronary heart disease. These

biomechanisms may help to explain why cigarette smokers have such an increased risk of developing coronary heart disease and of dying from it.

An increasing amount of evidence has been accumulated in the past few years relating the development of clinical cerebrovascular disease to cigarette smoking. Most of this information has come from mortality studies (*17*, *18*), both retrospective and prospective, which show that both male and female smokers of cigarettes under the age of 75, as compared to nonsmokers, have higher death rates from cerebrovascular disease designated as the underlying cause of death on their death certificates. This may be especially true for younger cigarette smokers age 45–54 where males had death rates about 50 percent higher than nonsmoking males, and females had death rates about 100 percent higher than nonsmoking females. Under age 75, mortality ratios for stroke increase as the number of cigarettes smoked increases. No association has been shown for those aged 75 and over.

The new epidemiological evidence, then, indicates that cigarette smoking may be more closely associated with cerebrovascular disease than previously indicated in the population between the ages of 45 and 74 years. If cerebrovascular thrombosis (thrombotic brain infarction) accounts for this association, it is possible that some of the considerations of how cigarette smoking may produce coronary thrombosis also apply to the pathogenesis of cerebrovascular disease. Further research is essential to understand the relationships which exist between cigarette smoking and cerebrovascular disease.

Additional epidemiological evidence from prospective mortality studies provides confirmation that cigarette smoking is associated with increased death rates from aortic aneurysm (nonsyphilitic), for both men and women. In one study of male smokers an increase in death rates was noted with increases in amount smoked.

HIGHLIGHTS OF CURRENT INFORMATION

1. Additional evidence not only confirms the fact that cigarette smokers have increased death rates from coronary heart disease, but also suggests how these deaths may be caused by cigarette smoking. There is an increasing convergence of many types of evidence concerning cigarette smoking and coronary heart disease which strongly suggests that cigarette smoking can cause death from coronary heart disease.

2. Cigarette smoking males have a higher coronary heart disease death rate than nonsmoking males. This death rate may, on the average, be 70 percent greater, and, in some, even 200 percent greater or

more in the presence of other known "risk factors" for coronary heart disease. Female cigarette smokers also have higher coronary heart disease death rates than do nonsmoking females, although not as high as that for males. In general, the death rates from this disease increase with amounts smoked. Cessation of cigarette smoking is followed by a reduction in the risk of dying from coronary heart disease when compared with the risk incurred by those who continue to smoke.

3. A greater frequency of advanced coronary arteriosclerosis is noted in male cigarette smokers, especially in those who smoke heavily.

4. Additional evidence strengthens the association between cigarette smoking and cerebrovascular disease, and suggests that some of the pathogenetic considerations pertinent to coronary heart disease may also apply to cerebrovascular disease.

Smoking and Chronic Bronchopulmonary Diseases (Non-Neoplastic)

Conclusions of the Surgeon General's 1964 Report

1. Cigarette smoking is the most important of the causes of chronic bronchitis in the United States, and increases the risk of dying from chronic bronchitis.
2. A relationship exists between pulmonary emphysema and cigarette smoking but it has not been established that the relationship is causal. The smoking of cigarettes is associated with an increased risk of dying from pulmonary emphysema.
3. For the bulk of the population of the United States, the importance of cigarette smoking as a cause of chronic bronchopulmonary disease is much greater than that of atmospheric pollution or occupational exposures.
4. Cough, sputum production, or the two combined are consistently more frequent among cigarette smokers than among nonsmokers.
5. Cigarette smoking is associated with a reduction in ventilatory function. Among males, cigarette smokers have a greater prevalence of breathlessness than nonsmokers.
6. Cigarette smoking does not appear to cause asthma.
7. Although death certification shows that cigarette smokers have a moderately increased risk of death from influenza and pneumonia, an association of cigarette smoking and infectious diseases is not otherwise substantiated.

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Additional evidence from the four major prospective studies indicates that cigarette smokers have a markedly increased risk of dying from chronic bronchitis and pulmonary emphysema. The range of risk varies for cigarette smokers between three and 20 times the mortality rates for nonsmokers, and depends in part on the total amount smoked and the age group studied. Female cigarette smokers have similarly increased mortality risks although somewhat lower than those for males. Cessation of cigarette smoking is followed by a lower mortality risk relative to those who continue to smoke. Generally, pipe

and cigar smokers are much less affected than cigarette smokers by these diseases.

Problems of nomenclature and diagnosis make satisfactory differentiation of chronic bronchitis from pulmonary emphysema difficult when considering the epidemiologic data. Nevertheless autopsy studies support the relationship between smoking and mortality. In addition, recent information from morbidity studies indicates that smoking is associated with symptoms of chronic bronchopulmonary disease. Even relatively young cigarette smokers show increased respiratory symptoms and decreased ventilatory function. Cessation of smoking is usually followed by improvement of these characteristics. Although some individuals may have an increased susceptibility to respiratory disease, studies of twin-pairs in Sweden (*4*, *5*, *6*, *14*)—in which one twin is a smoker and the other is not—show that those who smoke have a much greater frequency of respiratory symptoms and abnormalities of ventilatory function than do their nonsmoking twins. This demonstrates that cigarette smoking is of greater importance than hereditary and constitutional factors in the pathogenesis of chronic bronchopulmonary disease. Similarly, occupational exposures and air pollution may also cause respiratory disease, but cigarette smoking is of much greater importance.

Additional clinical and experimental laboratory evidence confirms the fact that constituents in tobacco smoke are harmful to the bronchial mucosa of the respiratory tract. Bronchial changes have been produced in experimental animals exposed to cigarette smoke.

It is suspected that smoking has a direct toxic effect upon the alveolar tissue of human lungs, in which case this effect might be important in the pathogenesis of many though not all cases of human pulmonary emphysema. Additional indirect evidence exists to substantiate this suspected toxic effect, but additional research is needed to confirm or deny the presence of the effect. However, the presently available evidence (epidemiological, clinical, pathological, and experimental) strongly suggests that cigarette smoking may well play an important pathogenic role in many, although not necessarily all, cases of pulmonary emphysema. The fact that other causes of pulmonary emphysema exist does not detract from the validity of this inference.

Additional evidence strongly supports the conclusion in the Surgeon General's 1964 Report that cigarette smoking is the most important of the causes of chronic bronchitis in the United States, and increases the risk of dying from chronic bronchitis.

HIGHLIGHTS OF CURRENT INFORMATION

1. New data confirm and to some extent strengthen the conclusions of the Surgeon General's 1964 Report.

2. Cigarette smoking is the most important of the causes of chronic non-neoplastic bronchopulmonary diseases in the United States. It greatly increases the risk of dying not only from both chronic bronchitis but also from pulmonary emphysema.

3. Cessation of smoking is followed by a reduction in mortality from chronic bronchopulmonary disease relative to the mortality of those who continue to smoke.

4. Even relatively young cigarette smokers frequently have demonstrable respiratory symptoms and reduction in ventilatory function.

Smoking and Cancer

Conclusions of the Surgeon General's 1964 Report

Lung Cancer

1. Cigarette smoking is causally related to lung cancer in men; the magnitude of the effect of cigarette smoking far outweighs all other factors. The data for women, though less extensive, point in the same direction.
2. The risk of developing lung cancer increases with duration of smoking and the number of cigarettes smoked per day, and is diminished by discontinuing smoking.
3. The risk of developing cancer of the lung for the combined group of pipe smokers, cigar smokers, and pipe and cigar smokers is greater than for nonsmokers, but much less than for cigarette smokers. The data are insufficient to warrant a conclusion for each group individually.

Oral Cancer

1. The causal relationship of the smoking of pipes to the development of cancer of the lip appears to be established.
2. Although there are suggestions of relationships between cancer of other specific sites of the oral cavity and the several forms of tobacco use, their causal implications cannot at present be stated.

Laryngeal Cancer

Evaluation of the evidence leads to the judgment that cigarette smoking is a significant factor in the causation of laryngeal cancer in the male.

Esophageal Cancer

The evidence on the tobacco-esophageal cancer relationship supports the belief that an association exists. However, the data are not adequate to decide whether the relationship is causal.

Cancer of Urinary Bladder

Available data suggest an association between cigarette smoking and urinary bladder cancer in the male but are not sufficient to support judgment on the causal significance of this association.

Stomach Cancer

No relationship has been established between tobacco use and stomach cancer.

Additional chemical, experimental, pathological, and epidemiological evidence has been reported that substantiates the conclusions of the Surgeon General's 1964 Report concerning the various sites of cancer that were shown to be associated with or caused by smoking.

Lung Cancer

Deaths from lung cancer in the United States are continuing to rise rapidly. Epidemiological evidence concerning cigarette smoking and lung cancer has confirmed positive relationships with increasing numbers of cigarettes smoked, with increasing duration, and with decreasing age of initiation of the habit. Male cigarette smokers of less than one pack a day have mortality ratios as high as 10 and smokers of more than one pack a day have mortality ratios as high as 30.

There is a much smaller increase of the lung cancer death rates associated with pipe and/or cigar smoking than with cigarette smoking.

Additional evidence provides specific information on the increased mortality ratios of female cigarette smokers. These have significantly elevated mortality ratios ranging as high as 5 for the groups with greatest exposure. Lung cancer rates appear to be somewhat lower for women who have never smoked regularly than for men who have never smoked regularly. The mortality rates for women who smoke, although significantly higher than for nonsmokers, are lower than for men who smoke. How much of this is due to lower exposure to cigarettes and how much to other factors cannot be determined from the data available.

Ex-cigarette smokers are shown to have significantly lower death rates compared with those who continue to smoke. As discussed under the general topic of cessation earlier in this report, the finding of reduced lung cancer rates in the population of British physicians (*8*, *9*, *10*) over a period of time in which the proportion of cigarette smokers was dropping significantly can be interpreted as similar to a controlled cessation experiment and provides critical confirmation of the judgment that cigarette smoking is the major cause of lung cancer and that sharp reductions can occur in the risk from lung cancer with the cessation of smoking.

Additional information is available concerning the presence of known or suspected carcinogens in tobacco smoke. It has been reported that the "tar" and nicotine content of cigarette smoke* tends to reflect the tumorigenicity of this smoke, and that a reduction of the "tar" and

* The phrase " 'tar' and nicotine" is used here as a general indicator of total particulate matter in cigarette smoke.

nicotine content is accompanied by a reduction in the tumorigenicity. Research is needed to identify and separate the tumor-initiating and tumor-promoting agents in tobacco smoke and to elucidate their interactions in the pathogenesis of cancer. Similarly, while additional data are available concerning experimental carcinogenesis, it is not yet certain that the typical characteristics of human squamous-cell lung cancer, with invasion and metastasis, have been experimentally produced by tobacco smoke in animals. It should be noted that this may never be achieved not only because it may not be possible to duplicate man's smoking action for anatomic and physiologic reasons but also because of species' differences in cellular response.

There is evidence that certain other exposures, for example, occupational exposures to asbestos and uranium ore may interact with the cigarette effect to produce an enhancement of the tumor-producing effect. There is also information to indicate that the occurrence of second primary lung cancers in smokers may be more frequent than previously indicated.

Oral Cancer

Substantial mortality ratios are found for cancers of the buccal cavity and pharynx. Mortality ratios for cancer of the pharynx are especially high. There is some evidence implicating alcohol and/or dietary deficiencies in some of these sites. With the exception of the pipe-lip cancer relations there are too few cases related to the individual parts of the buccal cavity to evaluate each independently, and data are inadequate on the interaction of smoking with other factors. Although all forms of smoking have high mortality ratios with these sites, mortality ratios for those smoking cigarettes appear to be somewhat higher than for those smoking pipes and cigars, especially in the case of cancer of the pharynx.

Laryngeal Cancer

Continued evidence from the prospective studies supports the existence of a high laryngeal cancer mortality ratio for pipe and cigar smokers as well as for cigarette smokers. Data on the smoking habits of patients treated for buccal cancer subsequent to their therapy suggests that continuing to smoke after therapy may increase the likelihood of an independent laryngeal cancer. The epidemiological evidence supports the previous conclusion that cigarette smoking is a significant factor in the causation of cancer of the larynx.

Esophageal Cancer

Additional data from the prospective studies confirm the high mortality ratio previously found for smokers of all forms of tobacco.

Autopsy studies of smokers compared with nonsmokers specifically observing pathological changes in esophageal tissue have been reported from both smokers and nonsmokers who died from causes other than esophageal cancer. The findings were similar to the abnormalities generally accepted as representing premalignant tissue changes of the epithelium of the respiratory tract; that is, epithelial cells with atypical nuclei were found far more frequently in cigarette smokers than in nonsmokers. Tissue sections with basal cell hyperplasia were also found more frequently in cigarette smokers and, as with the atypical nuclei, these findings increased with amount of cigarette smoking. Additional data to evaluate the relative importance of smoking and alcohol, independently and jointly, would help clarify the significance of these findings.

Urinary Bladder Cancer

The Dorn (*13*) and the Hammond (*11*) studies both show mortality ratios over 2.0 for smokers of over 20 cigarettes a day, but the Doll-Hill study (*8*, *9*), based on only 38 deaths, shows no apparent relationship. Two retrospective studies have shown significantly higher proportions of smokers among patients than among controls. Small scale metabolic studies suggest that cigarette smoking may block the normal metabolism of tryptophan, which would lead to the accumulation of carcinogenic metabolites in the urine. Further studies to verify this finding and studies analyzing changes in the bladder tissue of smokers as compared with nonsmokers would be helpful in arriving at a judgment of the significance of the elevated death rates found in smokers in the largest of the prospective studies.

Stomach and Pancreatic Cancer

Epidemiological evidence does not show a significant relationship between smoking and stomach cancer. An association between cigarette smoking and pancreatic cancer is implied, but the significance of this association is not clear at the present time.

HIGHLIGHTS OF CURRENT INFORMATION

Lung Cancer

1. Additional epidemiological, pathological, and experimental data not only confirm the conclusions of the Surgeon General's 1964 Report regarding lung cancer in men but strengthen the causal relationship of smoking to lung cancer in women.

2. Cessation of cigarette smoking sharply reduces the risk of dying from lung cancer relative to the risk of those who continue.

3. Although additional experimental studies substantiate previous experimental data, additional research is needed to specify the tumor-initiating and tumor-promoting agents in tobacco smoke and to elucidate the basic mechanisms of the pathogenesis of lung cancer.

Laryngeal Cancer

The conclusion of the Surgeon General's 1964 Report that cigarette smoking is a significant factor in the causation of laryngeal cancer in the male is supported by additional epidemiological evidence.

Other Cancers

Additional evidence supports the conclusions of the Surgeon General's 1964 Report and indicates a strong association between various forms of smoking and cancers of the buccal cavity, pharynx, and esophagus. In the absence of further information concerning the interaction of smoking with other factors known or suspected as causative agents, further conclusions cannot be made at this time, although a causative relationship seems likely.

Additional epidemiological, clinical, and experimental data strengthen the association between cigarette smoking and cancer of the urinary bladder, but the presently available data are insufficient to infer that the relationship is causal.

Other Conditions and Areas of Research

Conclusions of the Surgeon General's 1964 Report

Peptic Ulcer

Epidemiological studies indicate an association between cigarette smoking and peptic ulcer which is greater for gastric than for duodenal ulcer.

Tobacco Amblyopia

Tobacco amblyopia [dimness of vision unexplained by an organic lesion] has been related to pipe and cigar smoking by clinical impressions. The association has not been substantiated by epidemiological or experimental studies.

Cirrhosis of the Liver

Increased mortality of smokers from cirrhosis of the liver has been shown in the prospective studies. The data are not sufficient to support a direct or casual association.

Maternal Smoking and Infant Birth Weight

Women who smoke cigarettes during pregnancy tend to have babies of lower birth weight. Information is lacking on the mechanism by which this decrease in birth weight is produced. It is not known whether this decrease in birth weight has any influence on the biological fitness of the newborn.

Psychosocial Aspects

The overwhelming evidence points to the conclusion that smoking—its beginning, habituation, and occasional discontinuation—is to a large extent psychologically and socially determined. This does not rule out physiological factors, especially in respect to habituation, nor the existence of predisposing constitutional or heredity factors.

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By and large the contributions to knowledge in this area of varied considerations have been meager, although a number of investigations

on one or another aspect of the problem of smoking and varied health consequences have been undertaken.

Peptic Ulcer

The relationship between cigarette smoking and death rates from peptic ulcer, especially gastric ulcer, is confirmed. In addition, morbidity data suggest a similar relationship exists with the prevalence of reported disease from this cause.

Tobacco Amblyopia

Tobacco amblyopia is now believed to be a manifestation of nutritional amblyopia, which is aggravated by the inhalation of tobacco smoke. Various vitamin B factor deficiencies may be involved and there is evidence to suggest that chronic low vitamin B_{12} levels may potentiate the toxic effects of cyanide in tobacco smoke.

Cirrhosis of the Liver

Increased mortality of smokers from cirrhosis of the liver is found in the prospective studies. This has generally been thought to be largely secondary to an association between smoking and heavy consumption of alcohol. Published data are inadequate to test this interpretation.

Maternal Smoking and Infant Birth Weight

Further studies have confirmed the fact that women who smoke during pregnancy tend to have babies of lower birth weight, but data are lacking to determine either the mechanism or the significance of this finding.

Psychosocial Aspects

There has been a sharp increase in the attention devoted to behavioral research since the Surgeon General's Report. A number of new concepts have been developed and more sophisticated multivariate approaches are being used. However, because of the recency of these studies very little in the way of findings has been published on which firm conclusions may be based.

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PART II

Technical Reports on the Relationship of Smoking to Specific Disease Categories

CHAPTER 1

Smoking and Cardiovascular Diseases

CONTENTS

SMOKING AND CORONARY HEART DISEASE

Coronary Heart Disease Mortality [1]

The relative importance of the association between cigarette smoking and coronary heart disease (CHD) as compared to the association of smoking with other diseases was previously described in the introduction to chapter 11 of the Surgeon General's 1964 Report.

In the United States more persons die from coronary heart disease than from any other single cause; and this most common form of fatal cardiovascular disease accounts for a greater percentage of excess deaths among cigarette smokers than do deaths from lung cancer. In 1964, there were 1,798,000 deaths from all causes, of which almost 545,500 or 30.3 percent, were due to atherosclerotic heart disease, including coronary heart disease. Table 1 gives the 1964 death rates for *coronary heart disease* per 100,000 persons by age and sex:

Table 1.—*1964 death rates for coronary heart disease per 100,000 persons by age and sex*

	All ages	25-34	35-44	45-54	55-64	65-74	75-84	85+
Both Sexes	285. 1	6. 9	53. 2	205. 8	576. 3	1, 384. 9	2, 957. 7	6, 882. 9
Males	354. 2	11. 0	90. 9	341. 3	889. 8	1, 942. 4	3, 623. 0	7, 409. 4
Females	218. 5	3. 0	17. 4	76. 8	286. 4	926. 5	2, 464. 0	6, 559. 0

Source: National Center for Health Statistics (*97*).

These data illustrate the dramatic increases in the risk of death from coronary heart disease as age advances. For males the rates among persons over the age of 45 appear to double from one decade to the next; among females the increased risk of death with advancing age is more dramatic—a threefold increase every 10 years. Of perhaps greater importance are the relatively low death rates among females, particularly below the age of 65, compared to males of comparable age. The mortality differential between the sexes becomes less as age advances; under 45 years of age the coronary death rate among men is five times as high as among women and in the 75–84 year age group it is only about 1.5 times as high.

The Surgeon General's 1964 Report determined a median mortality ratio (*99*) (pp. 109–110) for coronary heart disease of male current cigarette smokers of 1.7. Since this report, five large prospective

[1] All death rates throughout this chapter are per 100,000 population, unless otherwise indicated.

studies of smoking and mortality have been updated on the basis of longer periods of observation on each study subject. Current findings are therefore more definitive and permit more detailed analysis of the interrelationship of cigarette smoking to other significant variables such as age, sex, and the nature of the smoking habit in terms of amount and duration of smoking. Pertinent findings are presented below from the studies of veterans in the United States (*52*) and Canada (*14*) and the extensive data reported by Hammond (*47*), Doll and Hill (*25, 26, 27*), and Borhani (*17*).

The relative excess mortality associated with cigarette smoking is generally expressed in terms of a mortality ratio. This statistic is defined as the ratio of the number of observed deaths among smokers, to the expected deaths among smokers, if the age-specific mortality rates observed among non-smokers had prevailed (*52*). The process of computing the expected number of deaths among smokers takes into account and adjusts for any differences in the age distribution of the smokers and the nonsmokers under observation. Generally smokers are defined as persons currently smoking cigarettes, and non-smokers as those who never smoked or who never smoked regularly.

Table 2 shows the mortality ratios for coronary heart disease deaths among current cigarette smokers according to the amount smoked daily in U.S. and Canadian male veterans.

TABLE 2.—*Coronary heart disease mortality ratios, age-adjusted among current cigarette smokers by amount smoked daily*

	Cigarettes smoked daily				
	Under 10	10-20	21-39	More than 20	40+
U.S. male veterans	1.3	1.7	1.8	------	2.0
Canadian male veterans	1.6	1.6	------	1.8	------

SOURCE: U.S. veterans study (*52*) and Canadian pensioners study (*14*).

In both studies (*14, 52*) the mortality ratios were similar and increased with increasing intensity of cigarette smoking. Slightly higher ratios are reported in the U.S. veterans study for current smokers of cigarettes only.

The U.S. veterans study also permitted the comparison of age-specific coronary heart disease mortality rates for ex-smokers and current cigarette smokers (table 2A). From these data, it appears that cessation of cigarette smoking is followed by a reduction in risk of coronary heart disease mortality as compared to those who continue to smoke cigarettes.

TABLE 2A.—*Annual death rate per 100,000 from coronary heart disease by age, cigarette-smoking status and number of cigarettes smoked per day, U.S. veterans study*

Number smoked per day [1]	45–54		55–64		65–74	
	Current cigarette smokers	Ex-smokers [2]	Current cigarette smokers	Ex-smokers [2]	Current cigarette smokers	Ex-smokers [2]
1 to 9	195	125	594	432	1,374	1,105
10 to 20	297	133	830	557	1,577	1,260
21 to 39	390	57	912	743	1,701	1,366
40+	502	----	1,101	646	1,955	1,482

[1] This is the current rate of smoking for current cigarette smokers and the maximum rate attained for ex-cigarette smokers.
[2] Ex-smokers who stopped for reasons other than doctor's orders.

SOURCE: U.S. veterans study (*58*).

The Hammond study findings summarized in table 3 are based on coronary heart disease deaths reported over a 4-year period among approximately 1 million persons (441,000 men and 563,000 women).

TABLE 3.—*Coronary heart disease mortality ratios among current cigarette smokers only, by amount smoked daily*

Age and sex	Non-smokers	Cigarettes smoked daily			
		Under 10	10–19	20–39	40+
Men:					
45 to 54	1.0	2.4	3.1	3.1	3.4
55 to 64	1.0	1.5	1.9	2.0	2.1
65 to 74	1.0	1.3	1.6	1.6	(1)
75 to 84	1.0	1.2	1.4	1.1	----
Women:					
45 to 54	1.0	0.9	2.0	2.7	----
55 to 64	1.0	1.3	1.6	2.0	----
65 to 74	1.0	1.1	1.4	1.9	----
75 to 84	1.0	----	----	----	----

[1] Expected deaths were less than 10.

SOURCE: Hammond, E. C. (*47*).

Tables 3 and 4 show that both men and women who smoke cigarettes have relatively higher death rates from coronary heart disease than nonsmokers, although men have higher rates than women. For each sex and for each age group, the mortality ratios for coronary heart disease generally increase with increased intensity of cigarette smoking (table 3). The highest mortality ratios for both men and women are observed in the 45–54 year age-group; the coronary heart disease

death rates among heavy smokers in this age group are three times the death rates for nonsmokers for both sexes. The mortality ratios for both men and women decrease with advancing age in each intensity category. This trend may reflect the effects of selective survival of smokers who have survived the elevated risks at younger ages of coronary heart disease and other diseases associated with cigarette smoking.

Another explanation of the decrease in mortality ratios with aging is that the effect of smoking, while substantial in increasing death rates, cannot be expected to be proportionate to all other causes of coronary heart disease as age advances. Considering the advanced degree of atherosclerosis generally found among nonsmokers over age 65, the deleterious effect of smoking is more appropriately represented by the excess in death rates among smokers. Table 4 below shows the observed death rates from coronary heart disease among persons studied by Hammond and classified by age, sex, and smoking status. Although the mortality ratios decreased with age, differences in death rates, which reflect the numbers of persons who die in each age group, increase. This could be interpreted to mean that, although relative to other factors, the role of cigarette smoking tends to diminish with advancing age, the number of excess deaths per 100,000 smokers continues to rise with advancing age.

TABLE 4.—*Age-specific death rates from coronary heart disease per 100,000 persons by age, sex, and smoking status*

Age and sex	Smokers of cigarettes only	Nonsmokers	Excess rate smokers/nonsmokers [1]	Mortality ratio
Males:				
45 to 54	422	150	272	2.8
55 to 64	996	542	454	1.8
65 to 74	2,025	1,400	625	1.5
75 to 84	3,871	3,132	739	1.2
Females:				
45 to 54	66	33	33	2.0
55 to 64	275	163	112	1.7
65 to 74	941	653	288	1.4
75+	2,349	1,973	376	1.2

[1] Calculated from the data.

SOURCE: Hammond, E. C. [(47), p. 145.]

The relative decrease in death rates from coronary heart disease associated with the cessation of cigarette smoking is illustrated by table 4A.